Comments on the Long-Term Outcome of Schizophrenia

by John K. Wing

Abstract

Among the many technical issues dealt with in this symposium, two clinical problems are preeminent—how is schizophrenia defined and what is meant by outcome? Each problem is highly complex. Even given adequate designs, sampling, and analysis, the generalizability of the results depends on the extent to which the diagnostic and outcome criteria are independent of each other and reproducible. The authors amply demonstrate how far we are from achieving such comparability. Schizophrenia, at the moment, is diagnosable only on its manifestations, which can be influenced for better or worse by environmental conditions. Several standardized diagnostic systems are available but they recommend different sets of rules. It is premature, therefore, to speak of a "natural" long-term course. It is even doubtful whether further long-term studies (except perhaps of birth cohorts) should be attempted until more discriminating and reliable methods have been found. Short-term studies, however, focused on specific hypotheses, still hold out promise of yielding fruitful results.

My comments focus on two major clinical issues from among the many technical questions that are raised in the series of articles on long-term followup: (1) The outcome of what? (2) What is outcome? Both are fundamental for interpreting the results of longitudinal research and for the success of the authors' recommended strategies of hypothesis testing.

The two questions are addressed from radically different perspectives by Angst (1988) and McGlashan (1988). Each might well be regarded by the other as representing an approach typical of his continent. Angst (1988) writes in the context of European phenomenology—a "bottom-up" tradition that acts as a strong corrective to "top-down" typologies. Moreover, he does not need to start, as Harding (1988) does from the assumption that Kraepelin's (1910) pessimistic view of outcome still generally prevails and must be rebutted. Mayer-Gross (1932) had followed up 260 out of a total of 294 patients admitted to the Heidelberg clinic in 1912 and 1913. Sixteen years later, about 35 percent had made "social recoveries" and a further 5 percent were socially disabled but out of the hospital. In view of the very high death rate (43 percent of those who had remained hospitalized, probably due in large part to a near-starvation diet during World War I and its aftermath), this result could be regarded as, in itself, a substantial rebuttal. The three recent studies (first published in German) to which Angst (1988) refers suggest a better prognosis still.

Angst (1988) discusses the nosological status of paranoid, reactive, schizoaffective, and schizophasiform psychoses, and gives passing mention to the extensive subdivisions of Kleist and Leonhard (Leonhard 1979). He considers the wisdom of including a course criterion such as that in DSM-III (American Psychiatric Association 1980) and an age criterion such as most psychiatrists use without it being a formal part of nosology. He concludes, in effect, that the convenience of a broad clinical

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construct justifies its adoption but that, by the same token, it makes no sense to consider "schizophrenia" as a single disease entity. At a meeting to celebrate 60 years of the Heidelberg clinic where Kraepelin, Jaspers, and Kurt Schneider had worked, it was suggested that schizophrenia as a disease entity had been born in Heidelberg and that it had also died there. No one need mourn (Hafner et al. 1987, p. 42). More important, from the point of view of this collection of articles, Angst (1988) states categorically that it is hopeless to expect long-term studies to improve the diagnosis of schizophrenia.

By contrast, McGlashan (1988) surveys the North American scene where, until the time of the U.S.-U.K. Diagnostic Project (Cooper et al. 1972) and the International Pilot Study of Schizophrenia (IPSS) (World Health Organization 1973), nosological concepts were dominated by psychoanalytical theories. The introduction of the criteria of Feighner et al. (1972) started a reaction that culminated in a top-down rule system, the official DSM-III, becoming more or less universally used for research purposes. McGlashan (1988) suggests, on the basis of his survey, that these diagnostic systems do not constitute a group with uniform predictive validity. This could be interpreted as questioning the value of the operational diagnoses. If we accept evidence from the Iowa 500 and from Wynne et al. (1987) that the various versions of DSM select very different groups of patients (quite apart from the fact that most of the studies reviewed do not conform to the rigorous standards set for comparability by the two methods articles), it would seem reasonable to conclude, with Angst (1988), that no useful conclusion can be drawn.

Harding (1988), who bridges the Atlantic by comparing her own Vermont series with two European studies using eight types of course and numbers of predictor variables, comes to similar conclusions to McGlashan (1988). Both the sampling and the outcome criteria differ between the three series, and it is difficult to draw firm conclusions from the exercise other than that the course is variable. The article by Carpenter and Kirkpatrick (1988) introduces an ingenious technique for investigating the course of disorders, although defining the cross-sections and, in particular, defining onset (von Cranach et al. 1981), must give rise to problems. They also recommend the excellent strategy of studying the effect of negative and positive phenomena separately. Samson et al. (1988) provide a thorough survey of the international literature of schizophrenic or paranoid psychotic affective disorder and reach modest conclusions.

The Outcome of What?

To return to the first of my two questions, it may be worthwhile, in the face of a wide range of disagreement, to provide a broad bottom-up clinical information base in terms of phenomena that can be rated reliably to which a number of top-down rule systems can be applied. Otherwise there can be no guarantee that the phenomena have been assessed in a comparable way. Indeed, all the evidence points to substantial differences between schools and substantial variations even within the practice of one individual examiner. At the heart of such a system there must be a set of differential definitions of symptoms and signs. There is no need to begin again at the beginning when constructing such a system; there is a wealth of careful clinical observation to draw upon. Jaspers' (1963) General Psychopathology provided a good starting point for the Present State Examination (PSE-9)-CATEGO system's glossary (Wing et al. 1974), which is being updated for the successor system—SCAN/PSE10.

Such a data base yields profiles at several levels of abstraction so that the course can be examined from many clinical angles, in addition to the possibility of making different nosologies compete when testing hypotheses. The importance of this was illustrated in the two large international studies already mentioned. The most recent of several further-examples comes from a study of first admissions to hospitals serving Helsinki, Finland. Of those diagnosed as afflicted by schizophrenic or paranoid psychoses (in high concordance with CATEGO classes), a large proportion also had syndromes that, taken by themselves, would have resulted in a profile of diagnoses rather than a single one (Pakaslahti 1986).

When the whole clinical course is considered, the problem becomes even more complicated. It is compounded by the need to assess phenomena that are better regarded as impairments or disabilities than as symptoms (Wing 1967). Cognitive deficits are the best known example. The negative impairments, when severe, are relatively easy to recognize, but they pass imperceptibly into normality, and there is great scope for variation in deciding where the boundary should be drawn when applying diagnostic rules. Because they often precede the manifestation of productive symptoms, they can be regarded as abnormalities of personality, but this terminology.
needs to be used with great circumspection.

Negative phenomena, for example, can fluctuate in parallel with positive, can fluctuate independently, and can fluctuate in response to environmental events or pressures. These last factors can be different from the factors that precipitate florid relapse. Other cognitive factors also need to be taken into account, although they are rarely measured in followup studies. The profile of intellectual abilities and deficits is an obvious example, but more subtle characteristics of thought and perception are, as of now, difficult to measure reliably. The interactions between such factors are a matter for hypothesis and testing. The use of global terms like "personality disorder" tends to obscure their complexity without adding anything of theoretical value, while apparently more specific terms like schizoid or paranoid personality or schizotypy are still very fragile conceptually.

Further problems arise in the differential diagnosis between schizophrenia and disorders in the upper range of the autistic spectrum, which are not yet commonly recognized. Asperger's syndrome (Asperger 1944; Wing 1981), for example, is not mentioned in DSM-III-R (American Psychiatric Association 1987). The cognitive impairments characteristic of such conditions give rise to speech and behavior that are often misclassified as typically schizophrenic.

For all these reasons, a simple diagnosis, whatever the criteria used, will not provide a sufficient basis on which to mount a long-term followup study. I agree with Angst (1988) that such efforts cannot illuminate the nature of "schizophrenia."

**What Is Outcome?**

These difficulties in defining the subject of the followup study are paralleled by equivalent problems in measuring outcome. Nearly all long-term studies use indices compounded of both social and clinical criteria, thus making comparisons between surveys conducted in areas with different social characteristics very difficult. The problem is not easy to avoid even by restricting the criteria to what appear to be purely clinical items. It is possible partially to identify three kinds of components. The first is clinical and must be based on definitions that, as far as possible, do not include a social component (Wing 1978, pp. 22-23). This can be done with a degree of success, for example, in the case of Schneider's first rank symptoms (Schneider 1959), which should not be confused with culturally determined possession states (World Health Organization 1973, pp. 273-277). Such distinctions could not be perfect by any means, but there can be no excuse for not attempting to make them.

The second component can loosely be described as social disadvantage. A poor education, lack of training, little experience in exercising social skills, low level of intellectual ability, absence of social supports, lack of opportunity to exercise skills that are present, and lack of appropriate help once clinical disorder occurs can all lead to a poor social outcome irrespective of any contribution due to "schizophrenia."

The third component is less often considered but equally important. Whenever a disorder is characterized by frequent relapses or by long-term disability, self-attitudes, expectations for the future, and motivation to achieve a lifestyle acceptable in the individual's reference groups tend to change. Such changes often mirror the attitudes of important people in the afflicted person's social environment. In the limiting case, that environment may be one characterized, over long periods of time, by social stigma, deprivation, and disadvantage.

In summary, at least four elements can vary independently or together in contributing to the degree of social disability present at any particular cross-section of the course, that is, at successive outcomes. These are the productive symptoms, cognitive and negative impairments, social disadvantages (which may themselves be partially due to "premorbid" impairments), and adverse personal reactions. Any overall measurement is likely to be an amalgam of all four elements and cannot, therefore, be accepted as a measure of the natural history.

**Testing Hypotheses**

Whatever the underlying functional or structural abnormalities, there can be very little doubt that they involve mechanisms that mediate the interaction between host and environment. The long-term course will depend not only on the biological abnormalities, therefore, but on the environment encountered. In view of the complexity of the foregoing analysis, and the effort that needs to be put in even to achieve a modestly relevant battery of clinical and outcome measures, apart from the other technical complexities not under consideration here, it is doubtful whether further long-term studies to gain knowledge either about the nature of schizophrenia or about initial predictors of eventual outcome should be attempted.

Hypotheses concerning various
factors that influence outcome can be tested in other ways. As Breier (1988) points out, useful work can be done on quite a small scale. More powerful designs can also be used. The two World Health Organization studies mentioned by Lin and Kleinman (1988) show what can be done with a 5-year followup period. In fact, all the conclusions of the IPSS followup were demonstrated at the 2-year outcome. Studies of institutionalism and the effect of a poor social environment on negative impairments carried out in the late 1950's and early 1960's involved a wide variety of designs (Wing 1968). One entailed an 8-year followup of the long-stay schizophrenic population of three hospitals chosen because, initially, they provided very different milieus—thus taking advantage of a natural experiment (Wing and Brown 1970). Such work has important implications for the course since, as Carpenter and Kirkpatrick (1988) point out, the negative impairments have a strong, though by no means absolute, relationship to longer-term outcome. Such studies, together with work on the precipitants of acute relapse (e.g., too high therapeutic expectations, life events, emotional intrusion) have provided useful hypotheses to explain why the course in developing countries seems to be more benign than elsewhere. Both types of factors (those associated with negative impairments and those associated with relapse) can act rapidly, and recovery can also occur quickly. Interventions can be corrective in both types of cases (Wing 1968; Leff et al. 1985). But the factors can operate over long periods of time and can also cause attitudinal and motivational change that is more long lasting.

My conclusion remains that there is no such thing as a "natural" history of schizophrenia, both because schizophrenia can be described only in terms of its manifestations, which will continue to be variously interpreted until some of its elements are validated and perhaps turn out to have different outcomes, and because its course is in part determined by contingencies. Probably the best overall estimate for industrial countries of the long-term outcome of schizophrenia, defined rather more broadly than DSM-III standards and with no obligatory course component, but diagnosed by one expert and followed up by him, is that of Manfred Bleuler (1978). About 25 percent recover completely, with no need of further antipsychotic treatment. The symptoms of an intermediate group of about 50 percent continue to fluctuate over decades. Of the remaining 25 percent with more persistent problems, only about 10 percent suffer permanent lifelong invalidism. This group prognosis, Bleuler thinks, demonstrates an improvement during the first half of the century due to the introduction of better social conditions in the hospitals, with a further improvement later because of effective pharmacotherapy, but the proportions with the best and the worst outcomes have not changed.

I doubt whether, at the moment, a sophisticated research team could do much better than that.

References


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